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L3           12 DUPLICATE REMOVE L2 (7 DUPLICATES REMOVED)

=> d l3 1-12 bib ab

L3   ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2003 ACS on STN
AN   2003:360779  CAPLUS
DN   138:380400
TI   TAK1-TAB1 fusion protein: a novel constitutively
     active mitogen-activated protein kinase kinase kinase for use in drug
     screening
IN   Sugita, Naohisa; Sakurai, Hiroaki; Sato, Naoya
PA   Tanabe Seiyaku Co., Ltd., Japan
SO   Jpn. Kokai Tokkyo Koho, 34 pp.
     CODEN: JKXXAF
DT   Patent
LA   Japanese
FAN.CNT 1
     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
PI   JP 2003135070      A2   20030513       JP 2001-335988   20011101
PRAI JP 2001-335988           20011101
AB   A fusion protein comprising human transforming growth factor-.beta.-
     activated kinase 1 (TAK1) N-terminal MAPKKK
     domain and human TAK1 binding protein 1 (TAB1)
     C-terminal TAK1 activation domain, functional as active mutant
     TAK1, encoding cDNAs, recombinant expression, and use in screening TAK1
     inhibitors, are disclosed.  TAK1 and TAB1 are connect
     via a linker peptide.  Activation of JNK, p38, or IKK, or induction of
     cytokine prodn., such as IL-6, IL-1, or TNF, may be assayed for screening.
     TAK1 mitogen-activated protein kinase kinase kinase (MAP3K) is activated
```

by its specific activator, **TAK1**-binding protein 1 (**TAB1**). A constitutively active TAK1 mutant has not yet been generated due to the indispensable requirement of **TAB1** for **TAK1** kinase activity. In this study, the authors generated a novel constitutively active TAK1 by fusing its kinase domain to the minimal **TAK1**-activation domain of **TAB1**. Co-immunopptn. assay demonstrated that these domains interacted intra-molecularly. The **TAK1**-**TAB1** fusion protein showed a significant MAP3K activity in vitro and activated c-Jun N-terminal kinase/p38 MAPKs and I.kappa.B kinase in vivo, which was followed by increased prodn. of interleukin-6. These results indicate that the fusion protein is useful for characterizing the physiol. roles of the **TAK1**-**TAB1** complex.

L3 ANSWER 2 OF 12 USPATFULL on STN
AN 2003:232028 USPATFULL
TI Method of screening TGF-beta-inhibiting substances
IN Ono, Koichiro, Gotenba-shi, JAPAN
Ohtomo, Toshihiko, Gotenba-shi, JAPAN
Tsuchiya, Masayuki, Gotenba-shi, JAPAN
PA CHUGAI SEIYAKU KABUSHIKI KAISHA (non-U.S. corporation)
PI US 2003162228 A1 20030828
AI US 2003-384743 A1 20030311 (10)
RLI Division of Ser. No. US 2002-158895, filed on 3 Jun 2002, GRANTED, Pat. No. US 6551840 Continuation of Ser. No. US 2000-529279, filed on 11 Apr 2000, GRANTED, Pat. No. US 6451617 A 371 of International Ser. No. WO 1998-JP4796, filed on 22 Oct 1998, UNKNOWN
PRAI JP 1997-290188 19971022
DT Utility
FS APPLICATION
LREP FOLEY AND LARDNER, SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 4117
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for screening substances that inhibit binding between a **TAK1** polypeptide and a **TAB1** polypeptide, which comprises contacting the **TAB1** polypeptide to the **TAK1** polypeptide and a test sample and then detecting or determining the TAK1 polypeptide that is bound to the TAB1 polypeptide.

L3 ANSWER 3 OF 12 USPATFULL on STN
AN 2003:57486 USPATFULL
TI Novel protein TAB2
IN Matsumoto, Kunihiro, Aichi, JAPAN
PI US 2003040050 A1 20030227
AI US 2002-151569 A1 20020520 (10)
RLI Continuation-in-part of Ser. No. WO 1999-JP6466, filed on 19 Nov 1999, UNKNOWN
DT Utility
FS APPLICATION
LREP JANIS K. FRASER, PH.D., J.D., Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
CLMN Number of Claims: 62
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 3179
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel signal transducer TAB2 which acts as an adapter molecule of TRAF6 and TAK1 and mediates the activation of TAK1 in the signal transduction of IL-1 was isolated. TAB2 induced the activation of NF-.kappa.B and JNK by IL-1. The signal transduction by IL-1 was inhibited by inhibiting the signal transduction of TAB2 with the use of a dominant negative mutant of TAB2. A compound inhibiting the signal

transduction in TAB2 is useful as an anti-inflammatory drug.

L3 ANSWER 4 OF 12 USPATFULL on STN DUPLICATE 1
AN 2002:280188 USPATFULL
TI Method of screening TGF-beta-inhibiting substances
IN Ono, Koichiro, Gotenba-shi, JAPAN
Ohtomo, Toshihiko, Gotenba-shi, JAPAN
Tsuchiya, Masayuki, Gotenba-shi, JAPAN
PA CHUGAI SEIYAKU KABUSHIKI KAISHA (non-U.S. corporation)
PI US 2002155624 A1 20021024
US 6551840 B2 20030422
AI US 2002-158895 A1 20020603 (10)
RLI Continuation of Ser. No. US 2000-529279, filed on 11 Apr 2000, PENDING A 371 of International Ser. No. WO 1998-JP4796, filed on 22 Oct 1998, UNKNOWN
PRAI JP 1997-290188 19971022
DT Utility
FS APPLICATION
LREP FOLEY AND LARDNER, SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 4119
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for screening substances that inhibit binding between a **TAK1** polypeptide and a **TAB1** polypeptide, which comprises contacting the **TAB1** polypeptide to the **TAK1** polypeptide and a test sample and then detecting or determining the TAK1 polypeptide that is bound to the TAB1 polypeptide.

L3 ANSWER 5 OF 12 USPATFULL on STN
AN 2002:221385 USPATFULL
TI TAB1 protein and DNA coding therefore
IN Matsuomoto, Kunihiro, Nagoya-shi, JAPAN
Nishida, Eisuke, Kyoto-shi, JAPAN
PA CHUGAI SEIYAKI KABUSHIKI KAISHA (non-U.S. corporation)
PI US 2002119525 A1 20020829
AI US 2002-123427 A1 20020417 (10)
RLI Division of Ser. No. US 2000-688701, filed on 17 Oct 2000, ABANDONED Division of Ser. No. US 1999-406854, filed on 29 Sep 1999, GRANTED, Pat. No. US 6140042 Division of Ser. No. US 1996-752891, filed on 20 Nov 1996, GRANTED, Pat. No. US 5837819
PRAI JP 1996-300856 19961028
JP 1996-126282 19960424
DT Utility
FS APPLICATION
LREP Stephen A. Bent, Foley & Lardner, Washington Harbour, Suite 500, 3000 K Street, N.W., Washington, DC, 20007-5143
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1057
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L3 ANSWER 6 OF 12 USPATFULL on STN
AN 2002:238893 USPATFULL
TI Method of screening TGF-.beta. inhibitory substances
IN Ono, Koichiro, Gotenba, JAPAN
Ohtomo, Toshihiko, Gotenba, JAPAN
Tsuchiya, Masayuki, Gotenba, JAPAN
PA Chugai Seiyaku Kabushiki Kaisha, Tokyo, JAPAN (non-U.S. corporation)

PI US 6451617 B1 20020917
WO 9921010 19990429
AI US 2000-529279 20000411 (9)
WO 1998-JP4796 19981022
20000411 PCT 371 date
PRAI JP 1997-290188 19971022
DT Utility
FS GRANTED
EXNAM Primary Examiner: Whisenant, Ethan C.; Assistant Examiner: Lu, Frank W
LREP Foley & Lardner
CLMN Number of Claims: 50
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 4214
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for screening substances that inhibit binding between a **TAK1** polypeptide and a **TAB1** polypeptide, which comprises contacting the **TAB1** polypeptide to the **TAK1** polypeptide and a test sample and then detecting or determining the TAK1 polypeptide that is bound to the TAB1 polypeptide.

L3 ANSWER 7 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 2001:219578 BIOSIS
DN PREV200100219578
TI The MAPK kinase kinase TAK1 plays a central role in coupling the interleukin-1 receptor to both transcriptional and RNA-targeted mechanisms of gene regulation.
AU Holtmann, Helmut; Enninga, Jost; Kaelble, Solveig; Thiefes, Axel; Doerrie, Anneke; Broemer, Meike; Winzen, Reinhard; Wilhelm, Arno; Ninomiya-Tsuji, Jun; Matsumoto, Kunihiro; Resch, Klaus; Kracht, Michael [Reprint author]
CS Institute of Pharmacology, Medical School Hannover, Carl-Neuberg-Strasse 1, D-30625, Hannover, Germany
Kracht.Michael@MH-Hannover.de
SO Journal of Biological Chemistry, (February 2, 2001) Vol. 276, No. 5, pp. 3508-3516. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 9 May 2001
Last Updated on STN: 18 Feb 2002
AB Mechanisms of fulminant gene induction during an inflammatory response were investigated using expression of the chemoattractant cytokine interleukin-8 (IL-8) as a model. Recently we found that coordinate activation of NF-kappaB and c-Jun N-terminal protein kinase (JNK) is required for strong IL-8 transcription, whereas the p38 MAP kinase (MAPK) pathway stabilizes the IL-8 mRNA. It is unclear how these pathways are coupled to the receptor for IL-1, an important physiological inducer of IL-8. Expression of the MAP kinase kinase kinase (MAPKKK) TAK1 together with its coactivator TAB1 in HeLa cells activated all three pathways and was sufficient to induce IL-8 formation, NF-kappaB + JNK2-mediated transcription from a minimal IL-8 promoter, and p38 MAPK-mediated stabilization of a reporter mRNA containing IL-8-derived regulatory mRNA sequences. Expression of a kinase-inactive mutant of TAK1 largely blocked IL-1-induced transcription and mRNA stabilization, as well as formation of endogenous IL-8. Truncated **TAB1**, lacking the **TAK1** binding domain, or a **TAK1**-derived **peptide** containing a **TAK1** autoinhibitory domain were also efficient in inhibition. These data indicate that the previously described three-pathway model of IL-8 induction is operative in response to a physiological stimulus, IL-1, and that the MAPKKK TAK1 couples the IL-1 receptor to both transcriptional and RNA-targeted mechanisms mediated by the three pathways.

L3 ANSWER 8 OF 12 USPATFULL on STN

AN 2000:146088 USPATFULL
TI TAB1 protein and DNA coding therefore
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Chugai Seiyaku Kabushiki Kaisha, Tokyo, Japan (non-U.S. corporation)
PI US 6140042 20001031
AI US 1999-406854 19990929 (9)
RLI Division of Ser. No. US 1996-752891, filed on 20 Nov 1996, now patented, Pat. No. US 5837819
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028
DT Utility
FS Granted
EXNAM Primary Examiner: Schwartzman, Robert A.; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1108
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L3 ANSWER 9 OF 12 USPATFULL on STN
AN 1999:150965 USPATFULL
TI Tab1 protein and DNA coding therefor
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Chugai Seiyaku Kabushiki Kaisha, Tokyo, Japan (non-U.S. corporation)
PI US 5989862 19991123
AI US 1998-144178 19980831 (9)
RLI Division of Ser. No. US 1996-752891, filed on 20 Oct 1996, now patented, Pat. No. US 5837819
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028
DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1049
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L3 ANSWER 10 OF 12 WPINDEX COPYRIGHT 2003 THOMSON DERWENT on STN
AN 1999-312645 [26] WPINDEX
DNN N1999-233498 DNC C1999-092304
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder.
DC B04 D16 S03
IN OHTOMO, T; ONO, K; TSUCHIYA, M
PA (CHUS) CHUGAI SEIYAKU KK; (CHUS) CHUGAI PHARM CO LTD
CYC 83
PI WO 9921010 A1 19990429 (199926)* JA 195p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IS JP KE KG KR KZ LC LK LR LS LT LU LV MD MG

MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
US UZ VN YU ZW
AU 9896468 A 19990510 (199938)
EP 1043586 A1 20001011 (200052) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 11523715 X 20010410 (200128)
KR 2001031325 A 20010416 (200163)
US 6451617 B1 20020917 (200264)
AU 752461 B 20020919 (200272)
US 2002155624 A1 20021024 (200273)
US 6551840 B2 20030422 (200330)
US 2003162228 A1 20030828 (200357)

ADT WO 9921010 A1 WO 1998-JP4796 19981022; AU 9896468 A AU 1998-96468 19981022; EP 1043586 A1 EP 1998-950354 19981022, WO 1998-JP4796 19981022; JP 11523715 X WO 1998-JP4796 19981022, JP 1999-523715 19981022; KR 2001031325 A KR 2000-704319 20000421; US 6451617 B1 WO 1998-JP4796 19981022, US 2000-529279 20000411; AU 752461 B AU 1998-96468 19981022; US 2002155624 A1 Cont of WO 1998-JP4796 19981022, Cont of US 2000-529279 20000411, US 2002-158895 20020603; US 6551840 B2 Cont of WO 1998-JP4796 19981022, Cont of US 2000-529279 20000411, US 2002-158895 20020603; US 2003162228 A1 Cont of WO 1998-JP4796 19981022, Cont of US 2000-529279 20000411, Div ex US 2002-158895 20020603, US 2003-384743 20030311

FDT AU 9896468 A Based on WO 9921010; EP 1043586 A1 Based on WO 9921010; JP 11523715 X Based on WO 9921010; US 6451617 B1 Based on WO 9921010; AU 752461 B Previous Publ. AU 9896468, Based on WO 9921010; US 6551840 B2 Cont of US 6451617; US 2003162228 A1 Cont of US 6451617, Div ex US 6551840

PRAI JP 1997-290188 19971022

AB WO 9921010 A UPAB: 20030707

NOVELTY - A method of screening for substances which inhibit the binding of **TAK1** polypeptide to **TAB1** polypeptide comprises:

(a) contacting the polypeptide in the presence of a sample; and

(b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with **TAK1** or **TAB1** polypeptide first.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for substances obtained by the screening method.

ACTIVITY - None given.

MECHANISM OF ACTION - TGF- beta signal transmission inhibitor/activator; extracellular matrix protein production enhancement inhibitor/activator; cell proliferation prevention inhibitor/activator; monocyte migration inhibitor/activator; physiological activity induction inhibitor/activator; immunosuppression inhibitor/activator; amyloid beta protein precipitation inhibitor/activator; TGF- beta inhibitors.

USE - The TGF- beta inhibitory substances can be used in drugs for indications e.g. as TGF- beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity (all claimed).

L3 ANSWER 11 OF 12 USPATFULL on STN
AN 1998:144215 USPATFULL
TI TAB1 protein
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Ueno, Naoto, Sapporo, Japan (non-U.S. individual)
PI US 5837819 19981117
AI US 1996-752891 19961120 (8)
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028
DT Utility

FS Granted
EXNAM Primary Examiner: Eliott, George C.; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 910
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L3 ANSWER 12 OF 12 DGENE COPYRIGHT 2003 THOMSON DERWENT on STN
AN AAY09544 peptide DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
DESC Human **TAK1** 6xHis **peptide**.
AB A method has been developed for screening for substances which inhibit the binding of **TAK1** polypeptide to **TAB1** polypeptide.
The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with **TAK1** or **TAB1** polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a peptide from an example of the present invention.

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